CLINICAL EXPERIENCE WITH CARDIAC ASSISTANCE BY MEANS OF INTRAAORTIC PHASE-SHIFT BALLOON PUMPING

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If the primary pathophysiologic defect in cardiogenic shock after myocardial infarction is low cardiac output secondary to ischemic failure of the heart as a pump, then it is rational to consider mechanical methods of circulatory support for patients with this syndrome[1, 2].

Our experimental work with a mechanical method for circulatory support by means of intraaortic phase-shift balloon pumping was reported previously[3]. The results indicated that balloon pumping effectively supports the circulation and can be continued for prolonged periods without deleterious effects to either the pumping components or the experimental subject.

A clinical trial was therefore undertaken. In its initial phase, our primary goal was to develop the technique. This report summarizes this experience.

MATERIAL

Since balloon pumping was an experimental procedure, only patients in terminal cardiogenic shock due to myocardial infarction were considered for inclusion in the initial stages of trial. The diagnosis of myocardial infarction was based on clinical, electrocardiographic, and enzyme findings. Cardiogenic shock was defined, in the face of maximal treatment with vasoactive drugs, as a syndrome manifested by most of the following signs:

1. Systolic blood pressure below 80 mm. Hg;
2. Generalized sympathetic reaction;
3. Mixed venous O_2 saturation below 60%;
4. Cardiac output below normal (2.5 L./min.);
5. Pulmonary edema; and
6. Persistent oliguria.

Balloon pumping was considered only when the cardiologists attending the patients had agreed that medical therapy had been exhausted and that the outlook was very grave.

Fifteen patients meeting these criteria were included in the present series (Table I).

TABLE I

AGE, SEX AND CLINICAL CONDITION OF 15 PATIENTS IMMEDIATELY PRIOR TO PHASE-SHIFT BALLOON PUMPING

Case	Age,	Sex	EKG	B.P. mm. Hg	C.V.P. cm.H_2O	Sympathetic Reaction	Oliguria	Arrhythmias	Pulmonary Edema	Mental Confusion or Coma	Previous M.I.
1	45	F	Posterior wall M.I.*	U*	14	+*	+	-	-	+	-
2	58	M	Anterolateral wall M.I.	U	H*	+	+	+	+	+	+
3	66	F	Anterior wall M.I.	50/30	14	+	-	+	+	+	-
4	76	F	Anterolateral wall M.I.	U	5	+	+	-	+	+	-
5	48	M	Anterolateral wall M.I.	80/50#	14	+	+	-	+	+	+
6	79	F	Anterolateral wall M.I.	50/30#	20	+	+	+	+	+	-
7	57	M	Posterior wall M.I.	60/-	14	+	+	+	+	-	-
8	72	M	Posterolateral wall M.I.	30	30	+	+	A*	+	+	-
9	50	M	Anterior & posterior wall M.I.'s	U	H	+	+	-	+	+	-
10	64	M	Anteroseptal M.I.	80/54	15	+	+	+ A	+	-	+
11	76	M	Anterolateral wall M.I.	75/40#	26	+	+	+ A	+	+	-
12	76	M	Posterior wall M.I.	70/40	23	+	+	+	+	+	+
13	60	F	Anteroseptal wall M.I.	U	40	+	+	+	+	+	+
14	54	M	Anteroseptal wall M.I.	50/-	6	+	+	+	+	+	-
15	65	M	Anteroseptal & lateral M.I.	97/71#	20	+	+	+	+	-	+

*Abbreviations: M.I. = myocardial infarction; + = present; - = absent; U = blood pressure unobtainable; H = distended jugular vein, no exact C.V.P. available; A = prior cardiac arrest

#During (Levarterenol) infusion.

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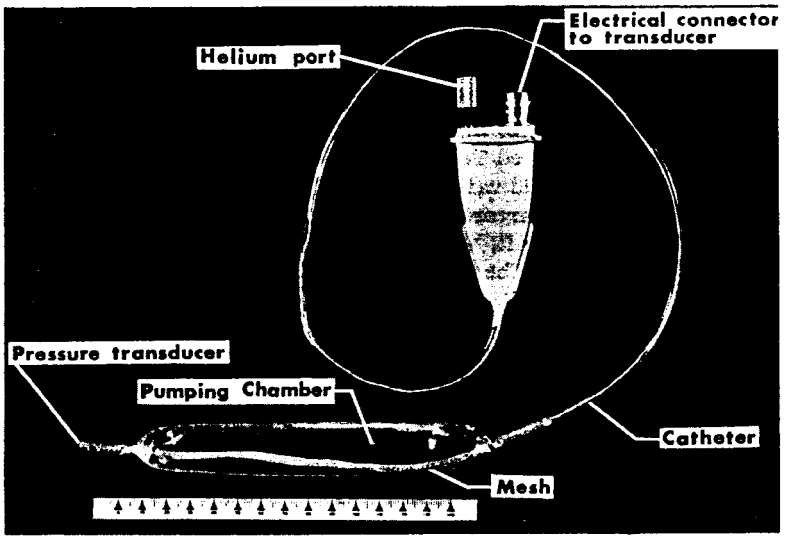

Figure 1. Intraaortic cardiac assistance balloon (human size).

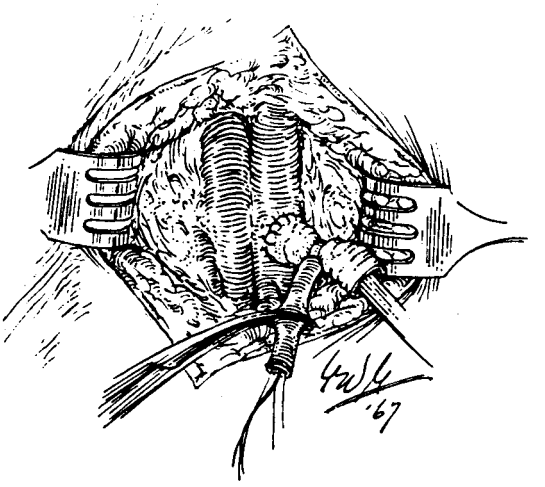

Figure 3. Dacron arterial graft in situ[5].

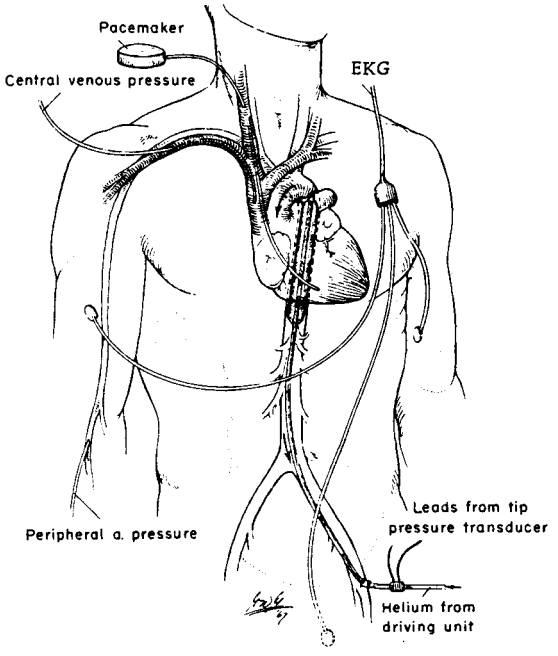

Figure 2. Schematic representation of balloon pumping set-up. Balloon shown in position for pumping[4].

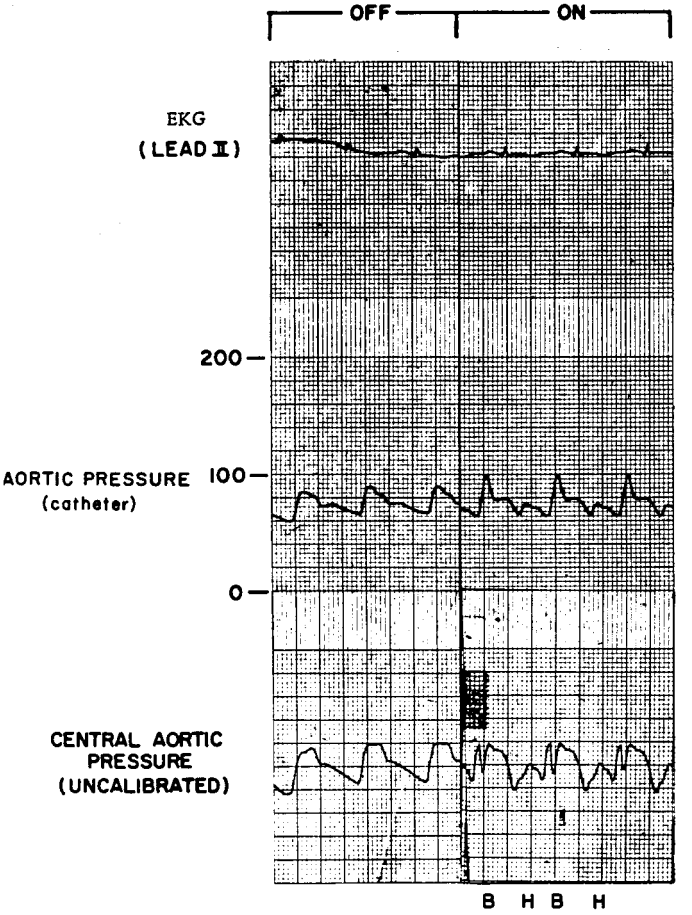

Figure 4. EKG and blood pressures with and without pumping. Bottom curve is derived from transducer incorporated in tip of pumping chamber. Note contributions of balloon (B) and heart (H). Contribution of heart is significantly reduced during pumping.

METHOD

The components of the balloon pumping system have been described previously[3, 4]. In brief, energy is introduced into the vascular tree during diastole of the cardiac cycle by inflation of a polyurethane pumping chamber with helium (Figure 1). A polyurethane catheter serves as helium conduit from an extracorporeal solenoid valve to the pumping chamber and carries leads from a pressure transducer, of the strain gauge type, embedded in the tip of the pumping chamber (Figure 2). To expand the balloon, a modified Tektronix Model 565 Oscilloscope is triggered by the R wave of the electrocardiogram to activate the solenoid valve.

The only surgery required for inititation of pumping is femoral arteriotomy, done at bedside under local anesthesia. The catheter is passed into the vessel through a Dacron graft, which then is sutured to the artery to permit restoration of the circulation to the extremity[5] (Figure 3). The pumping system is completely mobile.

All prior medications are discontinued, where feasible, at the initiation of pumping. Heparin, 50 to 75 mg., is given as the pumping chamber and catheter are introduced and at 4 hour intervals thereafter. When vasoconstrictive phenomena are present, chlorpromazine is given in small doses, 0.2 mg./Kg. body weight, to produce peripheral vasodilation and, thereby, to enhance the effectiveness of pumping. Cardiac arrhythmias are corrected pharmacologically or by means of a transvenous pacemaker. Correction of fluid and electrolyte abnormalties, mechanically assisted ventilation, and cardiotonic drugs are given as indicated.

Peripheral arterial and central venous pressures are monitored, as is the EKG; where possible, central aortic pressure is also monitored. The urine output is recorded. The arteriovenous O_2 difference and other blood gas and chemical parameters are periodically determined.

Balloon pumping is temporarily discontinued when the cardiogenic shock appears clinically resolved. If the circulation is maintained at adequate levels for several hours, the balloon is removed. If there are any indications of relapse into cardiogenic shock, however, pumping is resumed until stabilization is regained. At this point, pumping is again interrupted for a trial period.

RESULTS AND DISCUSSION

In all but one of the 15 patients treated, cardiogenic shock was clinically reversed during pumping (Table II). Early in the series, 2 patients died during temporary interruptions of pumping. In another patient, a signal from a demand pacemaker during a period when the pump was not in use led to irreversible ventricular fibrillation.

TABLE II

RESULTS OF PHASE-SHIFT BALLOON PUMPING IN 15 PATIENTS IN REFRACTORY CARDIOGENIC SHOCK

Case	Duration of Pumping*	Status
	Long-term Survivors	
1	4 hrs. 20 min.	Alive and well after 10 months
4	15 hrs. 37 min.	Alive and well after 6 months
7	5 hrs.	Alive and well after 5 months
9	10 hrs. 30 min.	Alive and well after 4 months
10	3 hrs. 30 min.	Alive and well after 4 months
14	14 hrs.	Alive and well afrer 1 1/2 months
	Short-term Survivors	
3	1 hr. 25 min.	Died 3 days later of CVA
5	10 hrs.	Died 7 days later of pneumonia and renal failure
8	55 hrs.	Died 3 days later from sudden rupture of mitral papillary muscle with acute ventricular failure
11	28 hrs. 30 min.	Died 8 hours later of cardiac arrest during sudden exertion
12	11 hrs.	Died 1 day later of confluent pneumonia
15	8 hrs.	Died 5 days later; no autopsy obtained
	Immediate Deaths	
2	1 hr. 26 min.	Died during interruption of pumping
6	3 hrs.	Died during interruption of pumping
13	25 hrs.	Died of fibrillation due to demand pacing during interruption of pumping

*Cumulative totals of periods of intermittent pumping

Six patients died 8 hours to 7 days after pumping was terminated (Table II). The third patient died 3 days after pumping from progression of a cerebral vascular accident. In Case 5, pulmonary and renal complications were responsible for the patient's demise a week after his recovery from cardiogenic shock. In Case 8, a ruptured papillary muscle of the mitral valve accounted for the patient's sudden death 3 days after pumping. The eleventh patient suddenly became agitated 8 hours after pumping, got out of bed, and expired. In Case 12, respiratory problems secondary to confluent pneumonia resulted in the patient's death 24 hours after the balloon was removed. The fifteenth patient, alert and responsive after pumping, had a temperature of 104° on the third day after pumping, became progressively obtunded, and died on the fifth day. It was not possible to obtain an autopsy.

Six patients were discharged from the hospital recovered from their infarctions and were well at the time of writing from 1 1/2 to 10 months later.

In its initial phase our investigation of intraaortic balloon pumping was intended primarily to determine whether this method of assisted circulation can support patients in medically refractory cardiogenic shock and to develop the method for use in patients. Our initial experience suggests that balloon pumping does have this capability.

It is also pertinent to consider the effect of balloon pumping on survival. It is generally agreed that the mortality rate in refractory cardiogenic shock is about 85 to 95%[6, 7]. In our series, 3 deaths occurred before the balloon was withdrawn from the patient's body (Table II). After pumping, the remaining 12 patients all maintained adequate circulation through their own cardiac action. Thus, 80% of the patients recovered from cardiogenic shock.

Eight other patients, who met the criteria for balloon pumping, died before permission to begin the procedure was given.

In all, 6 patients (40%) recovered from their infarctions and were well at the time of writing. Accordingly, there seem to be indications that the method compares favorably with medical modalities in cardiogenic shock.

Figure 4 represents a typical clinical recording of EKG and central aortic pressures before and during pumping. With the onset of pumping, the ventricular systolic pressure, as reflected by the aortic pressure wave, was significantly decreased. The time-tension index[8] in the 10 patients in whom it was possible to record central aortic pressures was reduced by an average of 25% (range, 11 to 50%), and mean aortic diastolic pressure was increased by 20%, on the average (range, 5 to 42%). In Figure 4 the portion labeled (B) represents the pressure wave exerted by the balloon's expansion. Note that this is approximately 180° out of phase with the normal cardiac cycle. For each cardiac cycle, pulsatile pressure to the periphery is increased and the frequency is doubled.

Because many of the patients were in extremis when pumping was initiated, it was possible only in 3 patients to obtain cardiac output determinations by the dye-dilution method before pumping. The values were as follows:

	Before Pumping	After Pumping
Case 5	2.5 L. /min.	4.8 L. /min.
Case 7	2.4 L. /min.	3.4 L. /min.
Case 12	1.5 L. /min.	3.8 L. /min.

In studies of dogs in experimental cardiogenic shock, coronary flow during balloon pumping was increased by 50%, on the average (range 39 to 65%). This suggests that increased coronary flow also occurs in patients as a result of balloon pumping.

Chlorpromazine was administered to nearly all patients. In laboratory studies, when this drug was given alone to animals in "severe" shock, mean diastolic pressure, and consequently coronary flow and cardiac output, usually fell. Initiation of pumping prevented the fall in diastolic pressure and increased coronary flow and cardiac output[9]. Thus, in addition to reduction in proximal aortic impedance attributed to the balloon, a further decrease in left ventricular load is produced by the vasodilator, chlorpromazine. This is associated in patients with a decreased tension-time index, which is related to myocardial O_2 consumption[8]. In this connection, the fact that it was possible to discontinue pumping in 12 patients suggests that left ventricular function was improved as a result of the mechanical-assist procedure.

Prior to initiation of pumping, pulmonary edema was present in 14 of the 15 patients (Table I), as evidenced grossly by pink frothy sputum or by pulmonary rales. When pumping began, these signs were reduced. At the same time, the decreased peripheral flow, as indicated by cold, clammy skin and absent distal pulses, was improved. Moreover, mechanical assistance made it possible to give large volumes (3 to 4 L. /24 hours) of intravenous fluids when necessary.

Increased urine output during pumping was observed in nearly all cases. Augmented peripheral flow resulting in increased renal blood flow was in all probability the major factor accounting for this. Prior administration of diuretics which had been ineffective because of hypotension, the administration of fluids made possible by pumping, and discontinuation of vasopressor medication were probably contributing factors. Data from ongoing patient trials should be helpful in elucidating these relationships.

During pumping, metabolic acidosis was corrected with sodium bicarbonate or THAM. Usually one dose of buffer was sufficient since pumping brought about good peripheral and central blood flow and marked diuresis. On the other hand, in patients managed medically, it is a common finding that repeated doses of buffers are needed to counteract the acidosis associated with cardiogenic shock.

The balloon pumping equipment functioned without failure in all patients. Pumping could be initiated within 20 to 30 minutes in most patients. In a few patients with obstruction of the left femoral artery, it was necessary to insert the balloon from the right side. In these instances the procedure was started within 40 to 50 minutes.

Hematologic studies did not disclose evidence of damage to the formed elements of the blood. In 5 cases, analysis of blood specimens obtained during pumping did not disclose hemoglobinemia, exhaustion of haptoglobin, significant fragmentation of erythrocytes, nor significant coagulopathies. Post mortem findings similarly did not reflect damage to the aorta or surrounding structures after pumping for as long as 55 hours, except in one case, in which a small subadventitial hematoma was found. In the first case, in which the Dacron arterial graft was not used, signs of arterial insufficiency were observed during pumping, because the distal circulation was occluded. In all subsequent cases, such complications have not been observed.

SUMMARY

1. Intraaortic phase-shift balloon pumping, a bedside procedure initiated under local anesthesia, was employed in 15 patients in terminal cardiogenic shock.

2. During pumping, signs of shock were reversed in 14 of the patients.

3. Twelve patients recovered clinically from shock, and it was possible to terminate the assist procedure.

4. Six of the patients died during the first week after pumping. Extracardiac causes accounted for or contributed substantially to these deaths.

5. At the time of writing, six patients were well 1 1/2 to 10 months after pumping.

6. Neither hematologic complications nor other damages due to pumping were observed. The pumping apparatus functioned without failure in all cases.

7. If further studies confirm our initial findings, balloon pumping may be a therapy of choice in refractory cardiogenic shock.

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